

Henry Van Wagoner

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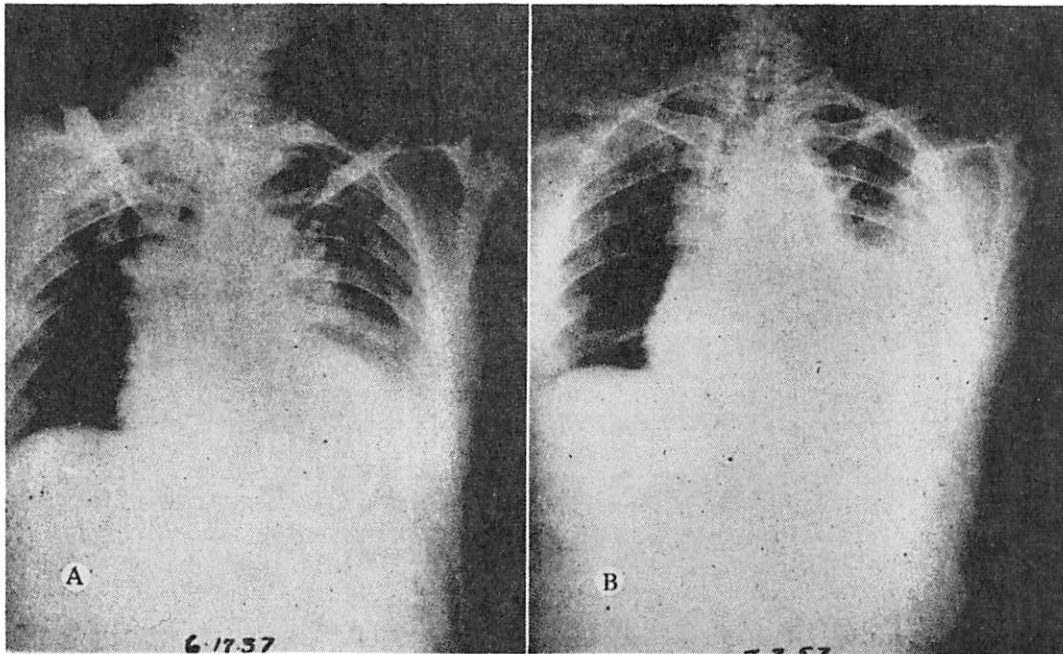


Fig. 8 (Case 4).—*A*, (bedside examination) on the third day after the coronary attack, there is evidence of left-sided pleural effusion. *B*, (bedside study, day of death) 23 days after *A*, left-sided pleural effusion has increased. Straightening of the upper half of the left border of the heart silhouette is compatible with pericardial effusion.

100.2 F, and for eighth days it continued at a level close to 100 F. The temperature was normal on the 12th and 13th days of illness.

On June 26 (the 13th day), the patient complained of severe pain in the midchest and left shoulder, which was aggravated by breathing. On the following day the temperature rose to 101 F and mild substernal pain was felt when the patient took a deep breath. A tentative diagnosis of pleuropericarditis was made. The temperature dropped to 100 F on June 28, 1957. A pericardial friction rub was first heard on July 1, the 18th day of illness, and remained audible for three days. The patient was then comfortable and felt only occasionally a tightening along the sternum when asked to take a deep breath. The white blood cell count at that time was 10,800 per cubic millimeter with 81% neutrophils.

Administration of bishydroxycoumarin was started on the day of the severe coronary attack. The prothrombin time was kept well within therapeutic range. During the first three weeks it was 30 seconds only once, and it remained below this level on all other days. Anticoagulant therapy was erroneously continued after the complication of pericarditis was diagnosed. Until July 6, frequent microscopic studies of the urine failed to show red blood cells; nor was there evidence of other hemorrhagic complications.

On July 5, the prothrombin time rose to 39 seconds; on the next day it was 33 seconds. How-

ever, "many red blood cells" appeared in the urine on that day. Anticoagulant therapy was then discontinued. The patient's condition gradually deteriorated. The temperature rose to 101 F on July 5 and to 102 F on July 7. X-ray studies on July 5 revealed an increase in the amount of fluid in the left pleural cavity; also, the possibility of pericardial effusion was mentioned.

On the morning of July 8, the heart rate was 128 and gallop rhythm was noted. The pulse was barely palpable. Percussion revealed increase of the cardiac dullness, which was thought to be due to pericardial effusion. A portable x-ray study (Fig. 8*B*) showed straightening of the left heart border, which was consistent with pericardial effusion. Also, the amount of fluid in the left pleural cavity appeared increased in comparison with previous studies. A clinical diagnosis of cardiac tamponade due to pericarditis and hemo-pericardium was made. Pericardial paracentesis yielded about 30 cc. of stark bloody fluid whose hematocrit was 33%, while that of the simultaneously tested venous blood was 38%. Since it proved difficult to withdraw a larger amount of pericardial fluid by aspiration and the patient's condition rapidly deteriorated, it was decided to perform pericardiectomy. During the preparations the patient died.

*Postmortem Examination.*—Examination revealed coronary arteriosclerosis with occlusion of the descending branch of the left coronary artery;